# Migraine without aura and ischemic stroke

Sir,

Migraine-related strokes have recently [5] been classified as: 1) coexisting stroke and migraine; 2) stroke with clinical features of migraine; 3) migraine-induced stroke; 4) stroke of uncertain classification.

We discuss the relations between migraine and ischemic stroke with reference to the case of a 35 year old woman with a long history of migraine without aura who, concomitantly with a worsening of headache, developed a right sensorimotor hemisyndrome after a hemispheric cerebral infarct. 7 months before consulting us, while on estrogen-progestogen therapy, the patient had had a bout of severe pulsatile headache of the migraine type lasting 20 days, followed by a right facio-brachiocrural sensorimotor hemi-syndrome together with aphasia. She has no diabetes or hypertension, does not smoke or use toxic substances. Indices of inflammation and blood coagulation and platelet tests were normal. There was no vasculitis. A two-dimensional echocardiogram revealed a slight mitral valve prolapse but no vegetation. A CT brainscan imaged several hypodense patches in the cortical and subcortical regions of the left hemisphere in the territory of the middle cerebral artery. Arteriography was normal. The patient was treated with aspirin (325 mg daily) and calcium antagonists.

The pathogenesis of migraine stroke includes vasospasm [4], neuronal depression [2] and/or increased platelet aggregation [3]. Stroke during an attack of migraine with aura is widely described in the literature and is considered to be migraine stroke. Some authors [1] have included migraine without aura in the classification of migraine stroke. At present, with the 1988 Classification of Headache Disorders, the relations between migraine and stroke and the criteria for the diagnosis of migraine are not well defined. This confronts the clinician with several difficulties in the classification and treatment of migraine stroke. In our view, the assessment is still more difficult when the patient has a history of migraine without aura and contemporaneously other factors that increase platelet aggregability and so heighten the risk of stroke. In such cases a stroke occurring in the course of an attack of migraine is hard to classify. This difficulty arises partly from the fact that it is still not clear whether the attack of migraine constitutes the etiopathogenetic moment or whether it is only a cofactor in triggering cerebral ischemia or whether it is simply a symptom of cerebral ischemia.

We consider that at the moment the prophylaxis of migraine-related stroke rests essentially on treatment to prevent attacks of migraine, especially in patients with risk factors for cerebral ischemia.

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